In the claims:

1. (Cancelled)

2. (Currently amended) ~~The~~ A peptide ~~of claim 1, wherein the~~ having an amino acid sequence ~~is~~ selected from the group consisting of:

YLTQPQS (SEQ ID NO. 1); and

TQLFPPQ (SEQ ID NO. 3).

3. (Currently amended) A peptide ~~up to 60 amino acids in length~~ comprising at least one amino acid sequence selected from the group consisting of:

a) YLTQPQS (SEQ ID NO. 1) or;

~~GSLPHSL (SEQ ID NO. 2);~~

TQLFPPQ (SEQ ID NO. 3);

~~HSIFDNI (SEQ ID NO. 4);~~

~~HHMPHDK (SEQ ID NO. 5);~~

~~YTTPPSP (SEQ ID NO. 6); and~~

~~QLPLMPR (SEQ ID NO. 7),~~

(b) a peptide up to 60 amino acids in length comprising the amino acid sequence of YLTQPQS (SEQ ID NO:1) or TQLFPPQ (SEQ ID NO: 3), wherein the peptide is capable of binding to Nog, MAG, and/or TN-R; and

(c) a peptide up to 60 amino acids in length comprising an amino acid sequences having at least 5 residues identical with corresponding residues in the amino acid sequence TQLFPPQ (SEQ ID NO: 3), wherein the peptide is capable of binding to MAG and/or TN-R~~, wherein the peptide is capable of binding to at least one of Nogo, MAG, TNR-EGFL and/or TN-R~~.

4. (Cancelled)

5. (Currently amended) A peptide up to 60 amino acids in

length comprising an amino acid sequence having at least 5 residues identical with corresponding residues in ~~at least one amino acid sequence selected from the group consisting of:~~

~~YLTQPQS (SEQ ID NO. 1);~~

~~GSLPHSL (SEQ ID NO. 2);~~

TQLFPPQ (SEQ ID NO. 3);

~~HSIFDNI (SEQ ID NO. 4);~~

~~HHMPHDK (SEQ ID NO. 5);~~

~~YTTPPSP (SEQ ID NO. 6); and~~

~~QLPLMPR (SEQ ID NO. 7),~~

wherein the peptide is capable of binding to ~~Nogo~~, MAG, TNR-EGFL and/or TN-R.

6. (Cancelled)

7. (Previously presented) The peptide of claim 5, wherein the number of identical residues is at least 6.

8. (Cancelled)

9. (Currently amended) The peptide of claim [[3]] 5, which is up to 40 amino acids in length.

10. (Currently amended) The peptide of claim [[9]] 5, which is up to 20 amino acids in length.

11. (Currently amended) The peptide of claim [[10]] 5, which is up to 10 amino acids in length.

12. (Currently amended) A composition for the treatment of CNS damage comprising one or more peptides ~~according to claim 1~~ selected from the group consisting of

(a) a peptide consisting of the amino acid sequence of YLTQPQS (SEQ ID NO:1) or TQLFPPQ (SEQ ID NO: 3);

(b) a peptide up to 60 amino acids in length comprising

the amino acid sequence of YLTQPQS (SEQ ID NO:1) or TQLFPPQ (SEQ ID NO: 3), wherein the peptide is capable of binding to Nog, MAG, and/or TN-R;

c) a peptide up to 60 amino acids in length comprising an amino acid sequence having at least 6 residues identical with corresponding residues in the amino acid sequence o YLTQPQS (SEQ ID NO:1), wherein the peptide is capable of binding to Nogo, Nogo66 or MAG; and

(d) a peptide up to 60 amino acids in length comprising an amino acid sequences having at least 5 or 6 residues identical with corresponding residues in the amino acid sequence TQLFPPQ (SEQ ID NO: 3), wherein the peptide is capable of binding to MAG, TNR-EGL and/or TN-R,

together with one or more pharmaceutically acceptable ingredients, said composition optionally being formulated for injection.

13. (Cancelled)

14. (Cancelled)

15. (Previously presented) A method for treating CNS damage in a patient in need thereof comprising administering an effective amount of the composition of claim 12 at or near a site of CNS damage in the patient.

16. (Previously presented) A method as claimed in claim 15, wherein said CNS damage is selected from the group consisting of spinal cord injury or stroke, said peptide has an amino acid sequence selected from the group consisting of:
YLTQPQS (SEQ ID NO. 1); and
TQLFPPQ (SEQ ID NO. 3), and is administered by direct injection into a site of spinal cord injury or stroke damage in the patient.

17. (Cancelled)

18. (Currently amended) A method of designing a mimetic of a peptide as defined in claim 3 [[1]], the mimetic having binding affinity for one or more of a neuronal growth inhibitory molecule selected from the group consisting of Nogo, MAG and/or TN-R, said method comprising:

(i) analysing a peptide of claim 1 that binds to one or more of said neuronal growth inhibitory molecules to determine the amino acid residues essential for the binding activity thereby defining a pharmacophore; and

(ii) modelling the pharmacophore thereby designing candidate mimetics, said method optionally comprising screening mimetics so designed for biological activity.

19. (Previously presented) The method of claim 18, which includes a step of assaying binding of a candidate mimetic to Nogo, MAG and/or TN-R in vitro.

20. (Previously presented) The method of claim 18 which includes a step, having identified a candidate mimetic that is capable of binding said neuronal growth inhibitory molecule in vitro, of optimizing the candidate mimetic for in vivo use.

21. (Previously presented) The method of claim 20, wherein the optimized mimetic is formulated together with one or more pharmaceutically acceptable ingredients.

22. (Currently amended) A bacteriophage which expresses at least one fusion protein consisting of at least one peptide of claim [[1]] 3 and a bacteriophage coat protein, such that the peptide is displayed on the surface of the bacteriophage virion.

23. (Currently amended) A screening method for identifying

peptides capable of binding to Nogo, MAG and/or TN-R, the method comprising:

providing bacteriophages of claim 22, ~~respectively~~ expressing said fusion protein consisting of said at least one ~~different~~ peptide~~s~~; and

screening the bacteriophages for the ability to bind to Nogo, MAG and/or TN-R.

24. (Previously presented) The method of claim 23, further comprising screening said bacteriophages or the peptides they display identified as binders for the ability to block the inhibitory effects of Nogo, MAG and/or TN-R on neuronal cell adhesion in an in vitro assay.

25. (Previously presented) The method of claim 24 further comprising formulating the peptide which blocks said inhibitory effects with one or more pharmaceutically acceptable ingredients for administration in vivo.

26. (Currently amended) A method of searching for factors which reduce the inhibitory effect of TN-R, MAG and/or Nogo, the method comprising interrogating a sequence database to identify polypeptides, or nucleic acids that encode polypeptide factors, that comprise an amino acid sequence having at least 5 residues identical with corresponding residues in an amino acid sequence selected from the group consisting of:

YLTQPQS (SEQ ID NO. 1); and

~~GSLPHSL (SEQ ID NO. 2);~~

TQLFPPQ (SEQ ID NO. 3);

~~HSIPDNI (SEQ ID NO. 4);~~

~~HHMPHDK (SEQ ID NO. 5);~~

~~YTTPPSP (SEQ ID NO. 6); and~~

~~QLPLMPR (SEQ ID NO. 7)~~

said method optionally comprising screening said factors

so identified for the ability to reduce the inhibitory effect of TN-R, MAG and/or Nogo on neuronal cell adhesion and formulating said inhibitory peptide factors with one or more pharmaceutically acceptable ingredients for administration in vivo.

27. (Currently amended) A method of searching for factors which reduce the inhibitory effect of TN-R, MAG and/or Nogo, the method comprising screening a cDNA library with an oligonucleotide probe which is capable of hybridising under stringent conditions with a nucleic acid sequence that encodes an amino acid sequence having at least 5 residues identical with corresponding residues in an amino acid sequence selected from the group consisting of:

YLTQPQS (SEQ ID NO. 1); and

~~GSLPHSL (SEQ ID NO. 2);~~

TQLFPPQ (SEQ ID NO. 3);

~~HSIPDNI (SEQ ID NO. 4);~~

~~HHMPHDK (SEQ ID NO. 5);~~

~~YTTPPSP (SEQ ID NO. 6); and~~

~~QLPLMPR (SEQ ID NO. 7)~~ said method optionally comprising screening said factors so identified for the ability to reduce the inhibitory effect of TN-R, MAG and/or Nogo on neuronal cell adhesion and formulating said inhibitory peptide factors with one or more pharmaceutically acceptable ingredients for administration in vivo.

Claims 28-64 (Cancelled)